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Case Report

The usefulness of nifekalant for activation mapping of premature beat-triggered atrial fibrillation: Suppression of atrial fibrillation initiation without inhibiting premature beat

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ABSTRACT

A 66-year-old man underwent a second ablation for atrial fibrillation (AF). Intravenous isoproterenol administration caused the atrial premature beat (APB), triggering AF. The APB originated in the right atrium and invariably initiated AF. Therefore, contact activation mapping could not be performed without frequent electrocardioversion. To prevent the initiation of AF without inhibiting the APB firing, we administered nifekalant intravenously, which facilitated precise activation mapping and ablation of the AF-triggering APB. The administration of nifekalant may improve clinical outcomes of catheter ablation for AF triggered by non-pulmonary vein APB, which invariably initiates AF.

1. Introduction

Ablation of the atrial premature beats (APBs) that trigger atrial fibrillation (AF) is an essential procedure for patients with AF. However, APBs originating from sites other than the pulmonary vein (PV) and superior vena cava are often difficult to eliminate. In some patients, these APBs invariably initiate AF, and frequent electrocardioversion is required for activation mapping. Here, we demonstrate a useful effect of nifekalant in a difficult case.

2. Case report

A 66-year-old man with a history of hypertension and symptomatic paroxysmal AF underwent extensive encircling PV isolation. However, after the initial successful PV isolation, he developed multiple episodes of paroxysmal AF after 3 months of blanking periods. Six months later, he underwent a second catheter ablation. After confirming electrical isolation of the 4PVs, intravenous isoproterenol (1.7 and 3.4 μg/min) was administered. This provoked frequent APBs originating from the right atrium (Fig. 1). These APBs invariably initiated a sustained AF. Therefore, contact activation mapping using the CARTO system (Biosense Webster Inc, Diamond Bar, California, USA) could not be performed without frequent electrocardioversion. To avoid the initiation of AF, we administered nifekalant (0.2 mg/kg bolus followed by continuous injection of 0.2 mg/kg/h) in addition to isoproterenol. This resulted in the maintenance of frequent APB firing without the initiation of AF (Fig. 2), allowing completion of activation mapping of both the sinus beat and APB, which was located 14 mm away from the sinus node at the right atrial free wall (Fig. 3). Radiofrequency ablation was delivered cautiously with an irrigated tip catheter at a power of 25 W for 45 s, which eliminated the APB. During the 6-month follow-up period, no AF recurrence or sinus node dysfunction was observed, and the patient was not taking any antiarrhythmic drugs.

3. Discussion

We successfully prevented the initiation of AF without inhibiting the AF-triggering APB, and eliminated the AF-triggering APB located close to the sinus node without damage to sinus node function. Nifekalant is an I_{Kr} antagonist that prolongs the duration of myocardial action potential and prevents the initiation and persistence of arrhythmias, including AF, by the mechanism of reentry and triggered activity [1]. However, it does not directly affect myocardial depolarization, and therefore does not decrease the frequency of APBs that are initiated by abnormal automaticity, such as isoproterenol-induced APB.

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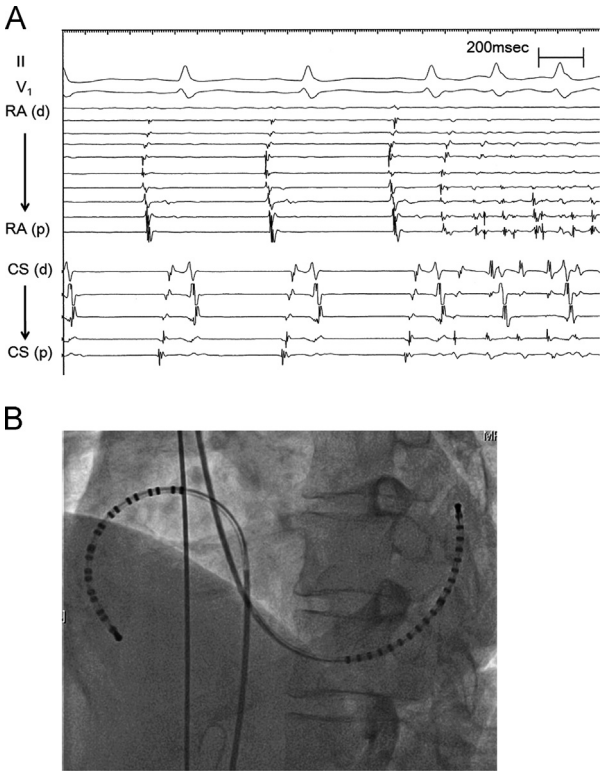

Fig. 1. (A) Atrial premature beat (APB) initiating atrial fibrillation (AF). Surface (II and V_1) and intracardiac electrograms from the right atrium (RA) and coronary sinus (CS) are shown. After administration of isoproterenol, frequent APBs originated from the RA. However, these APBs invariably deteriorated into sustained AF and activation map could not be performed. (B) Electrodes from the left anterior oblique projection. A decapolar deflectable catheter was positioned in the RA around the tricuspid annulus. Another decapolar deflectable catheter was positioned in the coronary sinus.

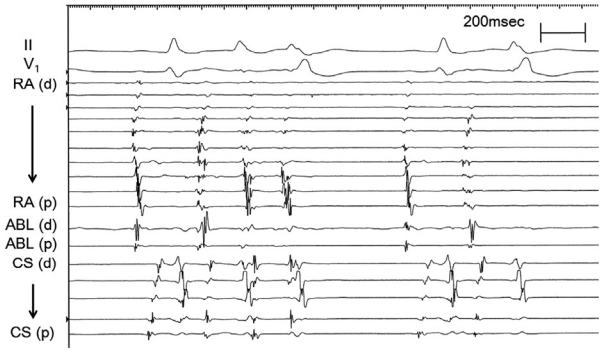

Fig. 2. Inhibition of atrial fibrillation (AF) initiation by nifekalant. Surface (II and V_1) and intracardiac electrograms from the right atrium (RA), ablation catheter (ABL) positioned at the earliest activation site in the RA, and the coronary sinus (CS) are shown. After administration of nifekalant in addition to isoproterenol, frequent atrial premature beat firing remained without initiating AF.

The successful elimination of non-PV AF-triggering APBs would improve the clinical outcome of AF ablation [2]. However, this can be challenging because APBs are rarely provoked, even after administration of isoproterenol. In addition, the administration of isoproterenol can easily result in the initiation of AF triggered by the APBs, which can hinder precise contact activation mapping.

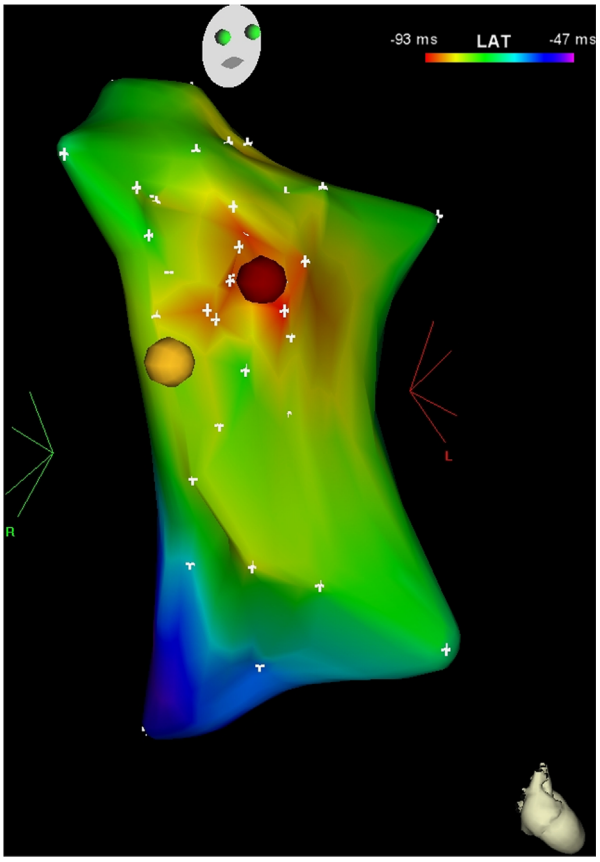

Fig. 3. The activation map of the AF-triggering APB on the CARTO system. This map demonstrated a centrifugal activation sequence with the earliest site at the right atrial free wall, 14 mm away from the sinus node (yellow tag). Radiofrequency ablation was delivered with an irrigated tip catheter at a power of 25 W for 45 s (red tag).

However, our observations can improve the clinical outcomes of ablation in patients with non-PV APBs, which initiate AF.

In summary, this case report describes that the administration of nifekalant during activation mapping of non-PV AF-triggering APBs prevented the initiation of AF without inhibiting APBs. Nifekalant may improve the clinical outcomes of catheter ablation for AF triggered by non-PV APBs, which invariably initiate AF. However, additional studies are required to determine whether this method is widely applicable.

Conflict of interest

None.

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None.

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